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Impact of sleep duration on seizure frequency in adults with epilepsy: A sleep diary study ☆

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ABSTRACT

Background: Prolonged sleep deprivation activates epileptiform EEG abnormalities and seizures in people with epilepsy. Few studies have addressed the effect of chronic partial sleep deprivation on seizure occurrence in populations with epilepsy. We tested the primary hypothesis that partial sleep deprivation over 24- and 72-hour periods increases seizure occurrence in adults with epilepsy.
Methods: Forty-four subjects completed a series of self-reported instruments, as well as 1-month sleep and seizure diaries, to characterize their sleep and quality of life. Diaries were used to determine the relationship between seizure occurrence and total sleep time 24 and 72 h before seizure occurrence using random effects models and a logistic regression model fit by generalized estimating equations.
Results: A total of 237 seizures were recorded during 1295 diary days, representing 5.5 ± 7.0 (mean ± SD) seizures per month. Random effects models for 24- and 72-hour total sleep times showed no clinically or statistically significant differences in the total sleep time between preseizure periods and seizure-free periods. The average 24-hour total sleep time during preseizure 24-hour periods was 8 min shorter than that during seizure-free periods ($p = 0.51$). The average 72-hour total sleep time during preseizure periods was 20 min longer than that during seizure-free periods ($p = 0.86$). The presence of triggers was a significant predictor of seizure occurrence, with stress/anxiety noted most often as a trigger. Mean total sleep time was 9 h, and subjects took an average of 12 ± 10 naps per month, having a mean duration of 1.9 ± 1.2 h. Daytime sleepiness, fatigue, and insomnia symptoms were commonly reported.
Conclusions: Small degrees of sleep loss were not associated with seizure occurrence in our sample of adults with epilepsy. Our results also include valuable observations of the altered sleep times and frequent napping habits of adults with refractory epilepsy and the potential contribution of these habits to quality of life and seizure control.

1. Introduction

Sleep deprivation is an emerging public health crisis. Estimates from the National Sleep Foundation in 2009 indicated that adult Americans sleep for only 6.7 h on average on weeknights and 7.1 h on weekends, and 12% report sleeping less than 6 h on worknights [1,2]. Sleep deprivation is associated with a variety of health and social consequences including increased all-cause mortality, cardiovascular disease, diabetes mellitus, obesity, hypertension, and respiratory diseases [3].

Sleep deprivation for 24 h or longer can lead to seizures even in individuals without epilepsy [4–8]. Sleep deprivation of this magnitude is often referred to as total sleep deprivation (TSD). In contrast, the role of partial sleep deprivation (PSD) as a seizure activator remains unknown. While two diary-based studies support the activating effect of PSD on seizure occurrence [9,10] and a significant percentage of patients with epilepsy subjectively report this as a trigger [11,12], a video electroencephalographic-based study found no relationship between PSD and seizure occurrence in adults with epilepsy in an epilepsy monitoring unit setting [13].

We, therefore, tested the hypothesis that PSD over 24- and 72-hour periods provokes seizures in adults with epilepsy. Then, we tested the hypothesis that lower habitual sleep duration (HSD) is associated with

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reduced health-related quality of life (HrQOL). In addition, we analyzed sleep diary data in order to describe sleep–wake patterns in our sample.

2. Material and methods

The study was approved by the Cleveland Clinic Institutional Review Board. Subjects provided informed consent prior to completing any research procedures.

2.1. Sample inclusion and exclusion criteria

Adults with epilepsy and a baseline seizure frequency excluding auras of at least 2 seizures per year (to increase chances of having seizures during the study duration) and not exceeding 1 seizure per day (to limit seizure clustering that might complicate analysis) were invited to participate. Patients who were unable to provide informed consent and comply with study procedures and those with unquantifiable seizures, history of substance abuse, and nonepileptic events that could not be reliably differentiated from epileptic seizures were excluded.

2.2. Data collection

Data were collected from a review of the electronic medical record including demographics (age, gender, height, weight, and smoking status), epilepsy characteristics (epilepsy type, seizure types, and mean monthly seizure frequency over 6 months prior to enrollment), antiepileptic drugs (AEDs) and daily dosage, and use of sedative hypnotics and psychotropic medications. Benzodiazepines were classified as used daily or intermittently. Epilepsy type was classified as focal or generalized, and seizure types were classified as generalized (convulsive or nonconvulsive) or focal (with or without impairment of consciousness) excluding auras.

Subjects completed the following instruments during their visits to the Epilepsy Center at the time of enrollment through the Cleveland Clinic Neurological Institute's Knowledge Program (KP), a computer tablet system for providing data to clinicians from patient-reported outcomes obtained prior to clinic visits:

1. Quality of Life in Epilepsy-10 (QOLIE-10) [14]: a 10-item questionnaire assessing HrQOL in adults with epilepsy;
2. Liverpool Seizure Severity Scale (LSSS) [15]: a 16-item scale with 2 subscales for seizure control and ictal/postictal effects;
3. Generalized Anxiety Disorder-7 (GAD-7) [16]: a 7-item validated instrument used to screen for anxiety disorder and to evaluate treatment outcomes;
4. Patient Health Questionnaire-9 (PHQ-9) [17]: a 9-item validated instrument used to screen for depression and to evaluate treatment outcomes;
5. EuroQOL 5D [18]: a 5-item questionnaire covering self-mobility, self-care, usual activity, pain/discomfort and anxiety/depression, as well as a visual analog scale (VAS) indicating overall well-being.

In addition, subjects completed the following sleep assessments:

1. Epworth Sleepiness Scale [19]: an 8-item survey ascertaining one's propensity to doze in common situations, in which a score of 10 or higher is considered abnormal;
2. Fatigue Severity Scale [20]: a 9-item tool for assessing fatigue using a 7-point Likert scale, in which a score of 36 or greater suggests significant fatigue;
3. Insomnia Severity Index [21]: a self-reported measure to evaluate perceived sleep difficulties, in which a score of 8 or greater suggests insomnia;
4. Sleep Apnea Subset of the Sleep Disorders Questionnaire (SA SDQ) [22]: a sleep apnea instrument with epilepsy-specific cutoffs of 26 or higher for women and 29 or higher for men [23] assessing the likelihood of having obstructive sleep apnea (OSA) based on variables including snoring, age, body mass index (BMI), tobacco use, and hypertension history.

Subjects were provided with a 1-month sleep and seizure diary and instructed on its use. On each day of data collection, subjects recorded the following:

1. Bedtime, wake time, time spent napping, and wake time after sleep onset (any time spent awake between listed bedtime and wake time), rounded to the nearest 30 min;
2. Presence or absence of seizure triggers including missed doses of AEDs, menstruation [24], more-than-usual stress [11,25,26], and alcohol use [27];
3. Seizures coded by type based on classification at the time of enrollment.

Total sleep time for each 24-hour period (from midnight to midnight of the following day) was computed from the diary. Total sleep time incorporated both night sleep and time spent napping while excluding wake time after sleep onset. Habitual sleep duration was the average total sleep time across the period of recorded diary days. A variable called "sleep deprivation" representing daily sleep changes was calculated as the difference between habitual sleep time and total sleep time for each 24-hour period recorded.

The presence or absence of any seizure triggers was recorded as an indicator value. Ideally, each trigger type would have been included individually, but many were noted for only a very small number of nights, so it would not be appropriate to comment regarding how these individually affect sleep or the chances of having seizures. This more conservative approach only identifies whether each night had something unusual that could affect sleep patterns and seizure activity.

Twenty-four-hour diary days were categorized as follows: seizure periods (days in which seizures occurred), preseizure periods (24-hour periods preceding a seizure period), and seizure-free periods (periods not preceding seizures or having seizures, see Fig. 1). Seventy-two-hour periods were similarly defined. Isolated auras were excluded.

2.3. Statistical analysis

Descriptive statistics were calculated for variables including age; gender; BMI; smoking status; and epilepsy characteristics including seizure types, seizure frequency, number of AEDs and AED standardized (STD) dose, scores on self-reported instruments, and summaries of sleep diary variables. The STD dose variable represents the amount of AED taken daily using the defined daily dose (DDD), a measure based on the assumed average daily dose in its main indication for adults assigned by the World Health Organization. Drugs taken as needed for prolonged seizures or seizure clusters were excluded. The prescribed daily dose (PDD)/DDD ratio was calculated and summed over all drugs for each subject. Standardized AED values >1 indicate dose regimens higher than the average.

Random effects models were used to compare total sleep time for seizure-free periods to sleep time during the 24- and 72-hour preseizure periods while accounting for correlations due to repeated measurements. Separately, a logistic regression model fit by generalized estimating equations was used to determine if sleep deprivation for over 24 h was associated with increased odds of seizure occurrence while controlling for age, gender, AED STD dose, and seizure triggers. Additionally, for the sake of consistency with existing literature, this logistic regression was repeated, with sleep deprivation coded as either present (at least 30 min of sleep deprivation that day) or absent, instead of treating it as a continuous variable.

To confirm that sleep times in well-rested patients did not mask a seizure-promoting effect of sleep deprivation in those with lower

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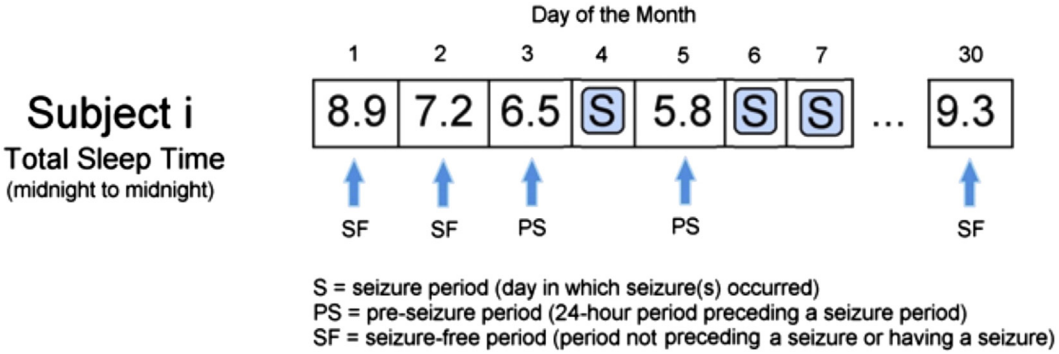

Fig. 1. Sleep data analysis diagram.

habitual sleep durations, we performed a sensitivity analysis, where the random effects analysis was repeated within the following two subsets of patients: low sleepers (those with sleep times below the first quartile of the sample) and high sleepers (those with sleep times above the third quartile of the sample).

Associations between HSD and HrQOL variables were explored using linear regression and ANOVA. For this analysis, HSD was categorized as ≤7.5, >7.5 to <9, and ≥9 h. Existing literature demonstrates adverse outcomes and increased mortality with <6 h and >9 h of habitual sleep [28–31]. However, only 2 subjects had HSD of <6 h, necessitating an increase of the lower limit to 7.5 h for a meaningful analysis.

Previous work suggests that a 30-minute reduction in sleep duration is associated with a 5% increase in the odds ratio of seizure occurrence [9]. Based on a reported 1.5-hour standard deviation for sleep duration [9], we estimated that 78 subjects would provide 80% power for detecting the effect of a 30-minute reduction in sleep on seizure frequency at a 0.05 significance level.

Results are presented as means ± SDs, unless otherwise indicated. $p < 0.05$ was considered statistically significant. Analysis was performed using R software (Vienna, Austria).

3. Results

3.1. Sample characteristics

Screening and enrollment are shown in Fig. 2. We screened 1328 patients, and 1108 did not meet the inclusion criteria, with the majority of them having seizures too infrequently. Among 220 patients meeting the inclusion criteria, 91 were directly contacted by the investigators, and 129 were sent recruitment letters. Fifty-five (60%) patients contacted by the investigators and 37 (29%) patients contacted by mail elected to participate. The total enrollment was 92, representing 42% of the eligible subjects. Ultimately, 44 (48%) of the enrolled subjects provided data for analysis. Reasons for attrition included withdrawal (lack of time/interest, N = 2; inability to accurately record seizures, N = 2; unplanned surgery, N = 1; poor health, N = 1; personal stressors, N = 1), loss to follow-up, and failure to return study materials. Patients who did not complete and return their diaries were similar in terms of age, gender, BMI, smoking status, epilepsy type, and LSSS scores to the subjects who completed the diary.

The mean age of the subjects who completed the series of self-reported instruments was 42 ± 13 years, and 10 (45.5%) were male. Mean BMI was 29 ± 8 kg/m^2, and 32% of the subjects were current or former smokers. Five (11%) subjects had generalized epilepsy, and 39 (89%) had focal epilepsy syndromes. Of the subjects with focal epilepsy, 6 (15%) reported focal seizures without impairment of consciousness (excluding auras). Thirty-one (79%) subjects reported focal seizures with impairment of consciousness, and 19 (49%) reported secondary generalization of focal seizures. All five subjects with generalized epilepsy reported convulsive seizures, including one with myoclonic seizures. One subject also reported nonconvulsive generalized seizures. The median monthly seizure frequency was 3 seizures per month. Ten (23%) subjects experienced less than one seizure per month. The mean score on the LSSS was 42 ± 26.

Subjects were taking a mean number of 2.3 ± 0.9 AEDs, with a mean STD dose of 3.5 ± 2.3 times the defined daily dose; nine (20%) subjects were on monotherapy. Nineteen (43%) subjects were taking sedative hypnotics (benzodiazepines or barbiturates) either regularly (7, 16%) or intermittently (11, 25%) for seizures. Nineteen (43%) subjects were taking psychotropic medications including antidepressants in 16 (selective serotonin reuptake inhibitors in 14 and tricyclic antidepressants in 2) and atypical antipsychotics in 4 subjects (one subject was taking both an SSRI and an antipsychotic). Seven (16%) subjects had comorbid sleep disorders, including three (7%) subjects with insomnia (2 taking sleep aids), three subjects with OSA (all using positive airway pressure therapy), and one (2%) with restless legs syndrome.

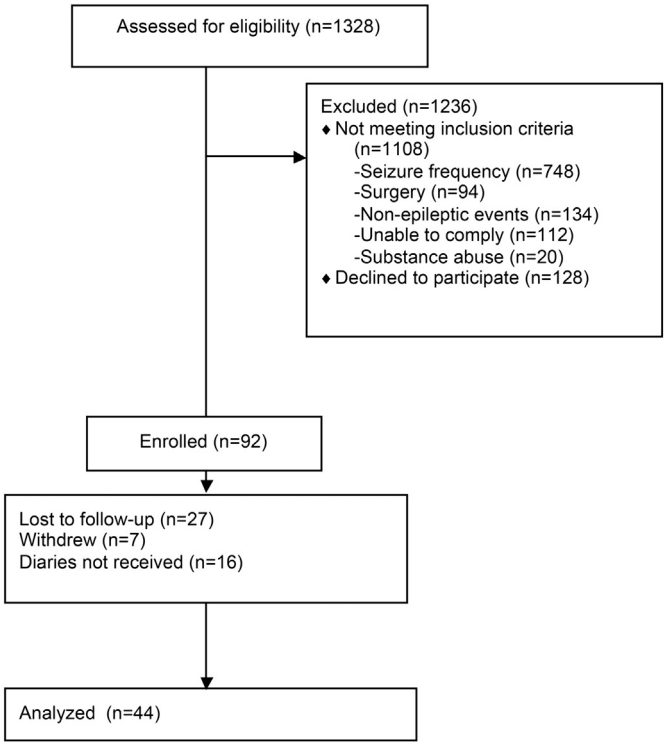

Fig. 2. Screening and enrollment flow diagram.

3.2. Self-reported instruments

The result of the sleep self-assessments suggested a high prevalence of sleep disorder symptoms. Mean score on the ISI was 9 ± 6, and 22 (50%) subjects had ISI scores above the cutoff, suggesting the presence of insomnia symptoms. Mean ESS score for the sample was 9 ± 5, and 10 (23%) subjects had elevated scores suggestive of excessive daytime sleepiness. Mean FSS score was 4 ± 2, and abnormal scores were obtained in 23 (52%) subjects. Four male subjects scored at least 29 on the SA SDQ, and 2 female subjects scored at least 26, for a total of 6 (14%) subjects with scores above the gender-specific cutoff for a positive sleep apnea screening. This included three subjects previously diagnosed with OSA. Mean SA SDQ score for the sample was 22 ± 6 for men and 17 ± 5 for women.

Similarly, self-reported mood and HrQOL data revealed impairments consistent with those observed in other adult populations with epilepsy. Mean PHQ-9 score was 7 ± 6, and 12 (35%) subjects reported abnormal levels of depressive symptoms, including 3 who endorsed suicidality. Ten (36%) subjects had elevated GAD-7 scores, with a mean score of 6 ± 7 for the sample. Mean self-reported overall health state based on the EuroQOL 5D VAS was 66 of 100 possible points. The mean QOLIE-10 score was 28 ± 8.2. Neither linear regression nor ANOVA analysis (Table 1) showed a significant association between HSD and EuroQOL or QOLIE scores, contrary to our hypothesis that longer or shorter sleep durations would correlate with lower quality of life.

3.3. Diary data

A total of 237 seizures were recorded during 1248 diary days, representing an average of 5.9 ± 7.5 seizures per month. Twelve (27.2%) subjects recorded no seizures during the 1-month data collection period. Only 1 of the 5 subjects with generalized epilepsy experienced seizures during the data collection period; this subject experienced 4 generalized tonic–clonic seizures. The 39 subjects with partial epilepsy experienced a total of 233 seizures, including 183 partial seizures and 50 generalized tonic–clonic seizures.

Mean HSD was 9.0 ± 1.8 h (4.7–15). Only 1 subject had HSD of <6 h, while HSD was >9 h in 16 subjects. The mean duration of the nighttime sleep period was 8.2 ± 2.3 h, naps making up the remainder of HSD. A total of 539 naps were recorded, 12 ± 10 per subject, having a mean duration of 1.9 ± 1.3 h. No relationship was observed between nap frequency and HSD or monthly seizure frequency. No relationships were observed between HSD and PHQ-9, GAD-7 scores, or AED STD dose.

Random effects models showed no statistically significant or clinically important differences in 24- or 72-hour total sleep times during preseizure periods and seizure-free periods. The average 24-hour total sleep time during preseizure 24-hour periods was 8 min shorter than that during seizure-free periods ($p = 0.51$). The average 72-hour total sleep time during preseizure periods was 20 min longer than that during seizure-free periods ($p = 0.86$). Similar findings were observed when excluding daytime naps from the total sleep time.

The sensitivity analysis, in which the random effects model was repeated using only the low sleepers (sleep duration below the first quartile of 7.92 h) and high sleepers (sleep duration above the third quartile of 9.58 h), showed similar conclusions. Neither subset showed evidence that sleep deprivation is associated with seizure occurrence ($p = 0.10$ and $p = 0.64$ in respective subset analyses).

Results of the logistic regression model are shown in Table 2. After controlling for age, gender, AED STD dose, presence of triggers, and baseline seizure frequency, we estimated the odds of having seizures in a particular 24-hour period to decrease (OR = 0.97), with increased sleep deprivation ($p = 0.57$). The only covariate having a significant effect on seizure occurrence was the presence of triggers (Tables 2 and 3). The most commonly reported trigger was stress/anxiety, with 40.1% of seizures occurring on days the subjects reported higher stress/anxiety than usual. When the logistic regression was repeated, with sleep deprivation (indicated by sleep time greater than 0/5 h but below average sleep on a particular night for a particular patient) as a predictor, results continued to show no significant increase in seizure occurrence after sleep deprivation (OR = 1.36, 95% CI: 0.93 to 2.01, $p = 0.10$).

4. Discussion

It is well accepted that profound sleep deprivation promotes seizures [5,6], and up to one-third of adults with epilepsy self-report sleep deprivation as a seizure trigger [11]. Nonetheless, our primary finding was that the sleep duration in the 24- and 72-hour periods before seizure occurrence did not differ significantly from HSD during seizure-free times. Our results, thus, suggest that mild day-to-day variations in sleep duration over a 1-month period do not necessarily promote seizures in adults with epilepsy.

Two prior studies evaluated the effect of sleep deprivation on epilepsy. In the first study, based on two-year sleep and seizure diaries completed by 14 adults with temporal lobe epilepsy, the probability of a seizure was significantly greater after a night of sleep deprivation compared with normal sleep [9]. The analyses in which we treated sleep deprivation as a continuous variable were designed to provide an estimate of the difference in sleep duration between the two different types of periods and to allow for higher sensitivity with limited sample size. There was, nonetheless, no association between sleep duration and seizures in our patients, whether sleep deprivation was treated as continuous or as categorical as done in these prior studies.

A more recent study involving 71 patients with focal epilepsy found that the odds of having a seizure the next day decreased for each hour of sleep the prior night [10]. This study included self-reported sleep time among its variables but did not include naps or wake time after sleep onset. Our methodology extends the previous work by including total sleep time and sleep deprivation before seizure and seizure-free periods and was, thus, designed to detect small contributions of sleep duration to seizure activation.

Variations in analytic methods; sleep duration definitions; and sample size, sleep time distribution, and disease severity preclude further direct comparison between our investigation and previous ones. Baseline seizure frequency was 5.9 seizures per month in our

Table 1
Health-related quality-of-life results by the sleep duration group.

Instrument	HSD (h)	N	Mean score	SE	p-Value
QOLIE-10	<7.5	6	26.0	3.4	0.74
	7.5–9	18	29.1	2.0	
	>9	9	28.7	2.8	
EuroQOL 5D VAS	<7.5	6	63.2	7.2	0.91
	7.5–9	18	66.7	4.1	
	>9	12	65.5	5.1	

HSD: habitual sleep duration; QOLIE-10: Quality of Life in Epilepsy-10; EuroQOL 5D VAS: visual analog scale portion of EuroQOL; and SE: standard error.

Table 2
Logistic regression model of seizure occurrence.

	Estimate	Odds ratio	p-Value
Age[a]	−0.01	0.99	0.41
Gender (female vs. male)	−0.17	0.85	0.64
AED STD dose	0.18	1.2	0.26
Triggers (yes vs. no)	0.57	1.77	0.01
Sleep deprivation (h)	−0.03	0.97	0.57

[a] Age centered at 40 years; AED STD dose: antiepileptic drug standardized dose; and sleep deprivation: daily difference between habitual sleep duration and total sleep duration.

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Table 3
Seizure triggers.

Trigger	Subjects (%)	Diary days (%)	Seizures (%)
AED noncompliance	9 (20.5)	38 (2.9)	6 (1.1)
Menses	12 (27.3)	91 (7.0)	11 (4.6)
Stress	34 (77.3)	330 (25.5)	95 (40.1)
Alcohol use	15 (34.1)	68 (5.3)	10 (4.2)

Number (%) of subjects, diary days, and seizures associated with seizure triggers from a total of 44, 1295, and 237, respectively.

subjects, and 75% of the subjects had >1 seizure per month. Furthermore, our patients took an average of 2.3 AEDs and had an AED STD dose of 3.5, indicating a high drug burden. Therefore, our findings may not be generalizable to populations with less pharmacoresistant seizures. A post hoc analysis did show, however, no significant association between habitual sleep time and seizure frequency during the monitoring period (data not shown). This suggests that seizure burden in patients with more refractory epilepsy did not inappropriately impact our analysis of sleep times.

The use of a detailed, standardized sleep diary allowed us to make additional observations about the unusual sleep and napping habits of our sample. The mean HSD of our subjects was 9.0 h, approximately 2 h longer than the major sleep period of the average adult American [1,2]. This observation not only could be related to the high antiepileptic drug doses in this population, to comorbid psychiatric diseases such as depression that may involve hypersomnia, or to error in self-reporting of sleep times but also may represent a particularly well-slept population. While our subgroup analysis suggests that the outcomes do not vary between groups with different baseline sleep durations, few subjects in the lower habitual sleep duration group slept for 6 h or less per night which has been associated with poor outcomes in the general population. We cannot assure that seizure occurrence in a group with even shorter habitual sleep times would not be more affected by sleep deprivation. Interestingly, the duration of the major sleep period was 8.1 h in one of the two prior diary-based studies in adults with epilepsy, which was similar to our results if naps were excluded [9]. No prior studies comment on daytime napping in populations with epilepsy. Naps contributed an average of 52 min to the mean daily sleep duration of our sample and occurred an average of 3 times per week for nearly 2 h per nap. If these findings are generalizable to the adult population with epilepsy at large, napping is an important component of daily sleep and must be considered when studying sleep times. We did not ask subjects why they took naps; however, self-reported daytime sleepiness and fatigue were prevalent and have been associated with impaired HrQOL in populations with sleep disorders and epilepsy [32–37]. Although not statistically significant, a positive correlation between HSD and HrQOL measures was found.

Many prior studies have investigated self-reported seizure triggers in populations with epilepsy, including but not limited to those cited above [11,12,26]. However, very few diary-based studies of seizures have incorporated such triggers into their analyses [10]. The inclusion of other commonly recognized seizure triggers is important to the study of the effects of sleep on seizure occurrence, as many of these triggers have an interaction with sleep and could be confounders. While the sample size and duration of our study limited our ability to include all commonly reported seizure triggers or to treat each trigger individually in our analysis, we felt that their inclusion was important in working to isolate the effects of sleep duration on seizure occurrence.

As in previous studies, half of our subjects reported symptoms of insomnia, but this seems to have gone largely undiagnosed, as only 14% carried a diagnosis and were actively treated for an insomnia disorder [32]. In addition, 52% of the subjects reported fatigue, and 23% reported excessive daytime sleepiness. Overall, the sleep pattern in our sample mimicked the disordered sleep pattern of other populations with chronic diseases, including those with cardiovascular disease, obesity, diabetes, and hypertension [1,38]. Factors that may impact sleep duration, sleep quality, and daytime symptoms in people with epilepsy include AED therapy, seizures, epilepsy type and localization, and comorbid sleep disorders. While we found no association between HSD and AED STD dose or seizure frequency, self-reported sleep disturbances are more common in people with epilepsy than in healthy populations [33,34,39,40]. Therefore, further investigation into the factors underlying sleep duration in epilepsy is warranted.

Sleep diaries are commonly used as adjuncts to the sleep history in populations with sleep disorders. However, their reliability remains unclear in populations with chronic disease. For example, a recent study of pharmacoresistant epilepsy found that patients believed that they spent more time sleeping than what was documented on polysomnography, a costly and labor-intensive procedure [41]. The difference between perceived sleep and actual sleep apparently resulted largely from sleep fragmentation, disturbed architecture, and the high prevalence of OSA in the 40 patients who participated. Approximately 30% of adults with epilepsy, regardless of seizure control, have OSA, which can adversely impact seizure control in some cases [42].

People with epilepsy may not document as many as one-half of their seizures even when being reminded daily to log them, presumably due to postictal confusion [35]. As with any patient-reported outcome, we cannot confirm the accuracy of seizure and sleep duration recording in our study. Also, for the purposes of analysis, aligning sleep times of the previous night to each seizure occurrence would have been preferable, but our data collection method did not include reliably recorded seizure times, so sleep data were instead collected in the same fashion as seizure data (by 24-h period) for consistency. Our results are also limited by the short duration of our study, which limited the number of seizures observed and our ability to detect effects of long-term sleep disruption, and defined the minimum baseline seizure frequency of our recruits. In addition, only half of the patients who enrolled in our study returned completed materials. The extent to which responders and nonresponders differ in terms of sleep and seizure control remains unknown.

5. Conclusions

Significant sleep deprivation represents an ominous public health burden when one considers its effects on social and occupational performance, health-care costs, and HrQOL and the potential for increased seizures in people with epilepsy. However, our results suggest that minor, day-to-day variations in sleep duration do not necessarily increase the risk for seizures in adults with epilepsy. Our study adds to the growing body of literature illustrating the spectrum of abnormal sleep habits and the high prevalence of sleep disorder symptoms in populations with epilepsy, further supporting the value of routine screening for common sleep disorders in epilepsy clinics.

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Conflicts of interest

None of the authors listed have any financial or other conflicts of interest to disclose with regard to this work.

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